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THOUGHTS ON ANGINA PECTORIS*

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HIGH or low, rich or poor, monarchists, republicans or communists, we poor human beings labour alike under the tyranny of words. To most of us, to a greater or less extent, certain terms, certain names, certain adjectives, the moment they are uttered, evoke pictures in our minds sometimes agreeable, sometimes repulsive, sometimes menacing, of such vividness and intensity that the words themselves, ambiguous though they may be, become to us in a sense entities. Such words, such phrases, may have an appalling influence on human action.

In medicine it is as in life in general. The influence of a mere clinical term may sometimes be considerable. Such a term, while it may describe but a group of clinical symptoms varying in physiological, anatomical or pathological import, comes to be regarded not only by the laity but too often by the profession, as such an entity. The mere term becomes, in our mind's eye, almost a living thing. Alas, to too many of us the essence is of less significance than the name. We are all more or less like the good woman who greeted my dear old master after a lecture on astronomy, and, congratulating him on his fascinating remarks, said: "But the most extraordinary thing, Mr. C., that which I can't understand, is how you discovered the names of the stars!"

POPULAR SIGNIFICANCE OF TERM "ANGINA PECTORIS"

"Angina pectoris"—what a picture these words evoke in the mind of the average man!—a picture of hopelessness, of agonizing suffering, of the constant menace of sudden death; a vague, indefinite apprehension of one of the most terrible fates imaginable. One of our vital duties as physicians is to deliver our patients from bondage such as that under which they labour, subjects to the tyranny of words such as these.

Not infrequently a patient in my consulting room says: "Doctor, is this angina pectoris?" In response I usually laugh and say: "Yes, if you will, it is 'angina pectoris.' But what is 'angina pectoris'? It is many things from a mere warning that you are growing older and that you mustn't be quite so active as you were twenty-five years ago—it is many things from this up to a really distressing and painful disease." And then I endeavour to enlarge upon this suggestion, pointing out to the patient the more hopeful side of the picture and dwelling upon the general management of his life until, usually, he leaves me calmed, encouraged, hopeful and ready, in so far as he is able to control himself, to lead the life that he ought to lead.

As a matter of fact "angina pectoris," as we use the words, is a term describing certain symptoms associated with cardiac and aortic disease—a syndrome which in itself varies widely in its manifestations and in its clinical course and prognosis. The anatomical alterations which are found post-mortem are generally associated with evidences of changes in the cardiac circulation and are, in my experience, less commonly due to aortic lesions other than those interfering with the coronary circulation than some of the modern literature would lead one to fancy.

WHAT DO WE MEAN BY "ANGINA PECTORIS"?

What do we generally include under the picture of angina or anginoid manifestations?

In a rough general way I should say:

1. Substernal pains or a sense of pressure or discomfort in the praecordium, brought on commonly by emotion or effort, sometimes by exposure to cold, always exaggerated by emotion or effort, always, if serious enough, necessitating the cessation of exercise or movement save in exceptional instances of which I shall speak. These sensations are associated generally with a radiation of pain or numbness or paraesthesia into the left arm more commonly, not infrequently into the right; into the neck, especially on the left side and, more rarely, into odd, distant localities. The first sensations of discomfort are very commonly in one or both arms, radiating to the substernal region. Pains in these localities brought on by effort or emotion and yielding with rest are always suspicious. I have seen angina in which, at the onset, the pains were referred purely to several teeth.

2. Severe spasmodic attacks coming on with emotion or apparently without cause which, save in the graver forms associated with coronary thrombosis, are relieved, almost always, temporarily, in their earlier stages by the nitrites.

As every physician knows perfectly well, anginoid sensations run all the way from the slight, tired, toothache-like feeling in the left arm or the

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indefinite sense of substernal pressure and discomfort, to the severe, vice-like, gripping, boring pains of the graver attacks. A rather characteristic feature of the paroxysm, especially in the more highly educated and sensitive, is the sense of apprehension that comes with it, and this, in itself, goes all the way from a simple realization that this is a warning signal to which one must pay attention, that he must stop, that he cannot really go on with what he is doing, to the intense *angor animi* and fear of death which is characteristic of the graver attacks. This condition is often associated with cutaneous hyperaesthesia or paraesthesia over the precordial area and upper chest and along the arms in the region of distribution of the last cervical and first two dorsal nerves.

Now it is quite obvious that pain in the distribution of a definite nerve supply may be caused not only by a referred pain as in these cardiac manifestations, but also by pressure on or by injury to the nerves themselves; and pain in that distribution common in angina is not so very infrequent in disease of the lower cervical or upper dorsal vertebrae, that which has given rise often to confusion. But here the conditions of onset of the pain and the nature of the attack are always different and a mistake should not be made. Cutaneous paraesthesia and hyperaesthesia in like regions occurs also in cardiac disease other than in angina.

DIAGNOSTIC REFLEXIONS

In recognizing the syndrome which we call angina pectoris those symptoms which are suggestive are not the mere character and distribution of pain, but the clinical course of the manifestations, the way in which the attacks come on, the manner in which they may be induced, the procedures by which they may be relieved, the way in which the patient behaves during the attack. Often, the age and physical conditions and surroundings and temperament of the patient, as every practitioner knows, play a part which may be conclusive. For instance, a girl of sixteen may complain of intense attacks of praecordial pain simulating angina very closely, and yet few of us would suspect that the manifestation was serious. We should demand confirmatory evidence of grave organic disease from the history, the physical signs or the results of other studies, that we might not feel necessary in a man of fifty; and we should usually find evidence enough that the attack was hysterical.

In like manner it is not at all uncommon to find in a young woman with a clean history, a story perhaps of abdominal pains, and a high degree of pulsation of the abdominal aorta; but we should not suspect an aneurysm. I have seen men rash enough to make a diagnosis of angina or abdominal aneurysm in such cases, but angina in a girl of sixteen or abdominal aneurysm in a young woman with good arteries elsewhere and without lues, are almost unheard of, and no one need give himself much anxiety under such circumstances unless the evidence is overwhelming.

One of the most important and characteristic features of angina is the appearance and behaviour of the patient during the attack. I shall never forget the picture of an old friend who, one day, I found on my doorstep, grey, pale, sweating, clinging to the railing, unable even to touch the button of the doorbell. This man, a few months before, had wanted to go to a well-known foreign bath resort for the treatment of cardiac disease. He was a native of the country in which this bath resort lay. I had warned him that if he decided to go he should first let me give him a letter to a distinguished clinician in that country; that if he went to the resort with a line from him he would be well cared for; otherwise he might easily receive a very careless sort of treatment—that which, alas, at that particular resort, was painfully common. He did not follow my advice. As he approached my house, boiling with indignation at the story he was about to tell me, his attack came on. When finally he was able to enter my consulting room his first words were: "I am ashamed of my countrymen." That picture of the fixed attitude, the pale, agonized expression, the ashen grey face covered with beads of sweat—that is the common picture.

One of the most pathetic instances that I remember was that of a man who, at the moment of the attack, was in the habit of rising from his bed, crossing the room to the mantlepiece on which he rested his left elbow, and stood swaying to and fro as he groaned gently, the tears pouring from his eyes, the sweat from his face—a distressing picture. Such attacks are uncommon, but are unmistakable when one sees them. Charles Sumner is said to have had the habit of walking about his room in severe attacks. But such movements are quite different from the violent muscular spasms of an hysterical attack.

Sometimes the relation of effort or emotion to the onset of anginoid pains may be entirely unappreciable to the patient. This is quite true in instances of coronary thrombosis. But after recovery, if recovery follow, the patient often appreciates the necessity of the restriction of physical effort and the relation of emotional strain to subsequent attacks of angina.

ANATOMICAL CHANGES IN ANGINA

But here let us stop for a minute and consider what we know about those anatomical changes which are associated with angina. At the very beginning, in the descriptions by Heberden and others, the calcified, narrowed coronary arteries were considered the most important elements in the picture. Since then much has been written about the frequency of coronary disease with angina, but many have laid emphasis on the circumstance which is undoubtedly true, that the gravest coronary disease, even thrombosis, may occur without anginoid pains. Others have called attention to the frequency with which the aorta shows signs of atheroma or syphilis. This has led some to feel that well-marked anginoid symptoms are rather more characteristic of aortic than coronary disease. Indeed, some are accustomed

to class as angina those attacks of nocturnal dyspnoea and anxiety so common in instances of syphilitic aortitis and aortic insufficiency. No one denies that coronary thrombosis may occur without much, or indeed perhaps without any of that which the patient actually describes as pain. No one denies that aortic disease may form the basis for anginoid attacks—for instance, by narrowing the mouths of the coronaries—but the more I see of angina the more I am inclined to feel that the picture of spasmodic attacks or discomfort induced by emotion or effort of the sort that I have described, is usually associated with coronary disease which interferes with the nourishment of the heart muscle and is, inferentially, often associated with painful coronary spasm. It may well be, as Keefer and Resnik [1] fancy, that the symptom is definitely associated with myocardial anoxemia. One of the most striking characteristics of anginoid pains is their relation to effort and emotion. But, as I have said, the immediate exciting cause of some of the sharp spasmodic attacks is hard to make out.

Those attacks, the gravest in their immediate import, which are associated with sudden coronary thrombosis, from which the patient recovers, are sometimes followed by years of disability in the sense that after the initial attack the patient finds himself in the same condition as does one in whom the onset of anginoid pains has been gradual; he can no longer take his accustomed physical exercise and he can no longer stand emotional strain without the appearance of anginoid pain. Here the symptoms have clearly followed a primary damage to the heart muscle by the coronary thrombosis.

The onset of mild anginoid symptoms, though commonly insidious, may then sometimes follow a definite coronary thrombosis. But it may also be sudden and without apparent cause, with the appearance, when the patient is at rest, of a slight aching pain perhaps in the substernal region or perhaps, at first, only paraesthesia or aching in one or both arms, pains which the patient may regard as rheumatic. Later, however, he finds that they are brought on or exaggerated by emotion or effort.

I think of such a patient whom I observed several years ago, a man in the early sixties who noticed, one evening, while getting ready for dinner, a rather uncomfortable "toothache-like" pain along the inner side of both arms. This individual, who was a physician, was rather struck by the location and character of the pain, which was unlike anything he had ever felt before. He avoided consulting his colleagues and kept very quiet for several days. He found out first that after several hours in bed, the pain disappeared, but recurred after rising. When it had entirely disappeared he found that unusual effort, such as brisk walking, brought the pains back immediately. Finally, after about a month, exceptional and unintended effort brought on an unmistakable attack of pain in the arms, radiating into the upper substernal region, which brought the subject to a standstill. With care this man has led a useful life since then, with very slow progress of his symptoms.

What happened to him when first he felt the pain? It seems to me that there is good reason to fancy that in such a case as this there was a sudden thrombosis of small terminal branch or branches of diseased coronaries. Up to the day of onset he had never noticed the least disability on exercise and he was a man who had taken rather violent exercise until the moment of the attack. The attack came out of a clear sky. Within a week or two afterwards tests showed that the characteristic disability had appeared.

Now in those cases of angina of gradual onset precipitated by emotion and effort, one usually finds either definite coronary disease or single or multiple areas of fibrosis in the heart muscle the cause of which is often not entirely clear, or both. I am rather inclined to think that time will show that in such cases as that just referred to, in which the onset, though very mild, is sudden and followed later by the symptoms characteristic of angina of effort—I am inclined to think that time will show that, in such cases, the onset has been associated with the occlusion of a small terminal branch or branches of the coronary vessels; not the brutal occlusion of a large branch with a considerable area of infarction of the heart muscle with its characteristic symptoms, but nevertheless a sudden thrombosis of final terminal branches which has produced enough interference with the circulation to bring on thereafter the characteristic symptoms of angina. I quite agree with my friend, Harlow Brooks, that in few instances of angina which one studies carefullv anatomically do we fail to find, at necropsy, rather definite coronary changes.

The answer of the opponents of the hypothesis that angina is usually associated with coronary disease—the answer, that many show coronary changes who have not had angina and that in some dead of angina, coronary disease has not been demonstrated—does not seem to me convincing. For coronary disease or multiple fibrous patches in the heart muscle are found in the great majority of instances, and the most characteristic picture of angina may be produced by coronary thrombosis.

EXCITING CAUSES

What then do we know about the cause of the syndrome which we call angina pectoris?

1. We know that the severe spasmodic attacks begin and run their course like spasms of involuntary muscle. We know that they are relieved in many instances by antispasmodics like the nitrites, which relax the arterial spasm. We know that in most instances the hearts of patients who have had attacks like this show obvious disease of the coronary vessels postmortem. We know that in those subject to angina, attacks may often be brought on or precipitated by emotion and effort.

2. We know that, in another sort of clinical picture, distressing sensations in these same regions and of the same character, though often

milder, may be produced by effort or emotion, yielding in the less severe instances, so soon as the effort is stopped. We know that in such patients the frequency of the paroxysms increases usually through the years. The attacks appear on less and less provocation until the wretched patient is bedridden. And we know that, at necropsy, there is generally either obvious disease of the coronaries or numerous sclerotic areas in the heart muscle not improbably the result of the gradual occlusion of terminal coronaries.

3. Finally we know that the most exquisite and persistent and unrelievable pain of exactly the same character, together with other suggestive symptoms of thrombosis, tachycardia, fall of pressure, fever, leukocytosis, may follow the occlusion of a branch of a coronary artery.

In other words, whatever justification there may be for other hypotheses as to the cause of angina pectoris in instances in which obvious disease of the coronaries has not been recorded, the evidence that it is related, for the most part, to coronary disease is very strong. We know that it may be brought on by coronary thrombosis; we know that, excepting by the use of morphia, the most satisfactory way in which to relieve it, save in coronary thrombosis, is by the use of the nitrites, which we know relax vascular spasm; and, in the third place, we know that evidence of actual disease of the larger or smaller coronaries, or occlusion of their mouths as a result of disease of the aorta, or evidence of disseminated fibroid patches in the heart muscle which mean the replacement of necrotic tissue which in many instances may best be accounted for by the hypothesis of the occlusion of terminal branches of the coronaries, are usually found at necropsy. These circumstances lead me to believe that the syndrome that we call angina pectoris is usually of coronary origin. That the character and distribution of pain in aortic disease—syphilis, aneurysms—is similar to that in anginal attacks is undoubted, but the spasmodic attacks of dyspnoea observed especially at night, usually seen in hypertensives, the "angina of rest" of Vaquez, form, it seems to me, a special, distinct picture. This picture I have not as a rule classed as "angina pectoris." I agree that in such cases evidence of coronary sclerosis or of fibroid changes in the heart muscle is not so common, though sometimes narrowing of the mouths of the coronaries and areas of fibroid change are found. So much has been written about coronary thrombosis in the last few years that it may be hardly worth while to enter into any lengthy discussion of the picture here. The history of the recognition of coronary thrombosis is, however, so interesting that I cannot refrain from saying a few words. I feel sure that had we not been so satisfied with the term "angina pectoris," had we been considering our patient from the proper standpoint, that is from the standpoint of one trying to make out physiologically what might produce these given symptoms, instead of being satisfied to classify them under a name, the clinical picture of coronary thrombosis would have been recognized many years before it was.

CORONARY DISEASE

Brought up with the feeling that was held by the old English authors that angina was usually a manifestation of coronary disease, it never occurred to me that the first instance of coronary thrombosis that I saw—in 1895—was anything other than a coronary thrombosis, and it never occurred to me that anyone else would have had any other view of the case. The patient was seen by Doctor Osler. We discussed it together. There was no necropsy, but I feel perfectly sure that he regarded it as an instance of coronary thrombosis as well as I. When I met with my second case in 1899, a most typical example, followed, two days later, by a pericardial rub, I recognized the case equally clearly, commented on it in my notes, and often talked about it to my students. I am perfectly sure that many physicians all over the world have recognized the syndrome in times past. The credit, however, of bringing the clinical picture before the medical public belongs to my dear friend, Herrick of Chicago, who first really called attention to it in 1906. It is truly extraordinary to see how many instances have been recorded since this time; how frequent a manifestation it is. As one of my distinguished colleagues observed the other day, it is perhaps too readily suspected by some. One might fancy that it was a new disease. How many new diseases are like coronary thrombosis, under our eyes every day of our lives but recognized by the world only when someone like Herrick has put the matter clearly before the public? I am always suspicious of new diseases.

I have spoken of the frequency of coronary disease, of the circumstance that coronary thrombosis followed by scarring of the area of infarction in the heart muscle and recovery may be followed by the development of characteristic anginoid pains on effort, and of the possibility that, in some cases, the sudden onset of mild anginoid symptoms without apparent cause, without the fever, leukocytosis, fall of pressure, tachycardia and other signs of an extensive infarction, may mean the sudden thrombosis of smaller terminal branches. Such an onset may be followed, at any rate, by the typical picture of permanent angina of effort. I have mentioned also that the symptoms of paroxysmal angina are, in their course, very like the spasm of smooth muscle fibre and that they are relieved by the nitrites, as if, in some way, disease of the coronaries or increasing demand on insufficient vessels brought on vascular spasm, though, of course, this is but a hypothesis.

One should not forget, however, the most interesting fact that, especially in hypertensives, beginning dilatation of the heart with evidences of pulmonary engorgement or particularly failure of the right side of the heart, not infrequently mark the end of anginoid pain. A patient who

for years has suffered from angina may lose his pains with the onset of congestive cardiac failure and, as Harlow Brooks has emphasized in a recent address, if coronary thrombosis may sometimes mark the beginning of anginoid pain, it sometimes, also, marks the end in that a large area of infarction upsets the cardiac compensation, and the patient dies after weeks or months or, indeed, years of congestive cardiac failure without the recurrence of angina.

Indeed sometimes an attack of coronary thrombosis, followed by symptomatic recovery, may be succeeded by a long remission in anginoid pains. This is due sometimes, I think, to the moral influence of the attack and the treatment which have impressed on the patient the necessity of leading a reasonable existence.

REFLEXIONS AS TO TREATMENT

But in this informal talk I want to dwell especially on the question of how we may help the sufferer from angina pectoris. Years ago, in speaking with my dear and wise old instructor, Dr. Frederick C. Shattuck of Boston, I observed that I always felt depressed and discouraged when I saw a patient with angina because there was so little that I could do. He laughed and said, in effect, that there were few conditions in which he felt he could do more. As the years have gone by I have come to realize fully how wise he was and how innocent and young I was. One can do much for many patients with angina; indeed the ability to help a patient with angina is a rather good test of the quality of the doctor. 'Tis a familiar truth and nowhere is it more apparent than in conditions such as this, that the wise physician accomplishes more by his kindly and intelligent advice and counsel than he does by his prescriptions and his medical treatment. The treatment itself varies greatly with the condition in which we find our patient, but under nearly all conditions the personal element, the tact, the judgment, the kindliness of the doctor, his willingness to take time to explain matters properly to his patient, to break unpleasant truths to him in such a way that he will look upon the hopeful side—these are often the most important elements of treatment. This applies equally to the family practitioner and the consultant. One cannot treat the patient with angina pectoris without giving him time and careful consideration.

Suppose a man comes to us, as he commonly does, when he begins to observe that effort produces unmistakable anginoid symptoms.

There is no more fascinating opportunity than that afforded by this situation, to relieve suffering and to prolong life; but it is a time-taking procedure. To begin with, to attempt to hide the nature of his condition from such a patient is silly, and certain to defeat our ends. Does that mean the necessary employment of the word "angina"? Of course not. The word "angina" is the very thing that we are seeking to avoid. We are trying to escape from the tyranny of alarming words, and to express the essence of the situation in such manner that it may encourage rather than depress the patient. In most instances this is quite possible to accomplish. But it demands time, time and careful explanation—explanation of the nature of the situation; that it is a warning, a red flag, and not a "smash-up"; that it is evidence of some defect in the circulation in his heart muscle; that it is the first notice which every man must have at one time or another, that, physically, he is not in the best condition; that every man of his age has some bad vessels; that many of us have the good fortune to have these in positions where they do no harm; that he, perhaps, has had bad luck, but that, after all, the warning may be rather a bit of good fortune than otherwise. And here I often refer to Osler's paper on "The Advantages of a Trace of Albumen and a Few Tube Casts in the Urine for a Man Over Fifty Years of Age," a diversion which often amuses and encourages the patient, at the same time impressing on him the truth. Or again I tell him that he is somewhat in the position of the patient with early tuberculosis, whose first symptom is an haemoptysis, often the most life-saving of incidents in that it draws attention to the existence of pulmonary mischief amenable to treatment, mischief which might otherwise be overlooked until too advanced for relief.

Here the value of experience becomes especially appreciable. We should preserve with the utmost care the records of the occasional medical miracles with which we all meet, and of the especially favorable cases in our practice. These will be among our most precious implements in the treatment of angina; they will be more valuable to us than most drugs. A true story of someone who has recovered from a like condition is often almost life-saving to the sufferer. He forgets everything else but the picture of that patient who recovered and soon, in his own heart, he comes to fancy that this perhaps may be the rule rather than the exception.

Only the most confirmed Christian Scientist exaggerates the importance of faith and hope in the practice of medicine.

In almost every instance of angina one is justified in encouraging the hope that if all goes well the patient may either recover entirely or at least be able, with certain reasonable modifications of his habits, to go on for a long period. It is a careless and sloppy method of practice to satisfy one's self by a few words with the patient, and by the statement that this is "false angina" and not "true angina." What we are trying to do is to escape from the dominion of terrifying and misleading words, and the words "false angina" produce in the patient's mind the picture of something as definite as his mistaken fancies concerning the meaning of "angina." Merely to give one's symptoms a name does not mean much. If one can make his patient feel that the word "angina" does not mean a sentence to suffering and death, but only describes a certain set of symptoms which vary enormously in their intensity and prognosis; that there is a considerable element of hope in his case, you can do far more for him. One must remember that the essential feature of our treat-

ment should be to encourage him to modify his life as he should; one can accomplish this only if the patient realizes the necessity.

And now after one has talked to him and encouraged him and led him to feel that what has happened may be hard luck, but not the end by any means, that it may indeed lengthen his life by inducing him to lead a proper sort of existence, after all this, what else have we that we can do for a patient with beginning anginoid symptoms? We can, it seems to me, do a great deal.

1. One must put the patient into the best possible physical condition. To do this we must carefully go over his manner of life. We must find out just what it is. Very often we find that he leads a disordered and hurried life. We must begin by inquiring into the character of his day, and these inquiries we must make not only of himself, but of his wife and others who observe him. We must see to it that he begins the day without hurry; that his habits are regular; that he takes plenty of time for his meals; that he eats deliberately and, of course, moderately; that he avoids constipation, and this is a matter often that needs the most careful attention and is very time-taking for the physician, for the treatment of constipation does not consist in simply prescribing a laxative. We must look carefully into his habits in view of the possibility that he may be subjected to some of the toxic influences which have been thought to play a part in inducing angina. Gout is certainly of importance. Tobacco may be of importance; it is certainly in instances of hypertension. While I, myself, have never seen an instance of angina which was definitely "cured," if one may use the word, by the omission of tobacco, I am sure that I have seen great benefit in some cases of nervous, heavy smokers, from the abandonment or modification of smoking. If the patient be one of those unfortunate, weak-minded invertebrates, of whom there are too many in the world, who "simply can't stop smoking," who cannot refrain from making himself a nuisance to his fellow man by standing around, red-eyed and "frowsy" headed, while he smokes his cigarette in the crowded dressing room of a sleeping car before he can begin his morning toilet, there is but one thing for him to do, and that is to stop it. Every man of that sort has a serious drug habit. If he is obviously smoking too much, and is a man, he should learn to smoke in moderation and only at leisure after his meals.

Every effort must be made to induce the patient to avoid hurry. A hurried day is often initiated by habits of rising and dressing in a few minutes. Some patients, if taught to realize this, may learn to add a quarter or even a half an hour to their dressing time, to read the paper during the hours of dressing, and arrange matters in such a way that, the initial hurry avoided, the day goes on with a calm with which they have been previously quite unfamiliar.

In order to put one's patient in the best possible condition the importance of searching for and relieving focal infections cannot be exaggerated. It is often impossible to say that the relief of this oral sepsis or that chronic prostatitis has been the cause of so much improvement, but there is no doubt whatever that occasionally the influence of focal infections, apparently unimportant, is far-reaching. I have had one instance of the disappearance of an angina following a tonsillectomy for good cause. The improvement, of course, may have been *post hoc* rather than *propter hoc*. However that may be, the incident is true, and so worth heeding, while from a therapeutic standpoint this experience has been of considerable value in helping me to induce patients to do what it seemed to me they should.

I am very apt to end my conversation with a patient of this sort by reference again to Osler's habit of speaking of the advantages of a trace of albumen in the urine for a man over fifty. "But," one may say, "suppose this man ask you about sudden death?" That is a bugaboo which, with most patients, is dealt with very easily. It is not the patient who is annoyed about that; it is the family. To the patient who asks you it is easy and true to say that he has a somewhat better chance than the average man of dying the most blessed sort of a death. That, alas, is about all, because many sufferers from angina die in other ways. Too many, alas, go through the distressing stages of progressive myocardial failure. It is not hard as a rule to make one's patient look at the possibility of sudden death as a blessing rather than a menace.

The medical treatment of such a patient, beyond special emergencies, is symptomatic. If the patient be syphilitic he has, of course, a door of hope, but syphilis is not the common basis of angina. In syphilitics it is exceedingly important to begin treatment with mercury and iodides, and not to use intravenous arsenical treatment until later. I have not happened, myself, to see sudden death follow the abrupt use of arsphenamine, but I have seen what seemed to me grave, immediate reactions.

The treatment of constipation I have already referred to. The treatment of the attacks may be summarized in two words—"nitrites, morphia." The nitrites often produce the desired result. It is only in the grave spasms that morphia is necessary when, of course, it should be employed freely.

I feel, as does Harlow Brooks, that either tablet triturates of nitroglycerine or liquid tincture of glonoin are the best forms in which to employ the nitrites. They are usually as good as nitrite of amyl. The nitrites should be employed symptomatically. Continued employment seems to me quite useless. The dose may be increased as is necessary. It is a great relief to many individuals to feel that they have in their pockets a ready relief of this sort. Other drugs, of course, help, but the nitroglycerine is so much simpler. Still one must not forget that it is very hard to make any absolute rule in medicine, and sometimes, where nitroglycerine, even in small doses, brings on uncomfortable flushing, other preparations such as Hoffman's anodyne or sweet spirits of nitre may help.

I have a dear friend who always carries in his pocket a lovely cut-glass cornucopia-shaped receptacle with a silver top—a receptacle which must have been intended, I should think, for smelling salts. This receptacle contains about two ounces of *spiritus frumenti*. A little straight whiskey stops the attack and the patient who, beside being a temperate man, is one of the most distinguished of our colleagues, ought to know. There are some advocates of temperance who call themselves Christians who might disapprove of this; but there is no intemperance more blind or more cruel, no immorality more pernicious than that practised by some well-meaning fanatics in the name of temperance and morality.

If the patient be hypertensive or obese these conditions must be considered and properly combated.

2. If the attacks become more frequent or, of course, if one find his patient in an attack suggesting a coronary thrombosis, or indeed, if, in a progressive angina, the signs of myocardial failure come on, then the urgent need is for rest—a long rest. What is the value of rest? In an acute cardiac infarction or with a myocardial insufficiency the value of rest is obvious. By saving every heart beat the heart muscle is given an opportunity to regain strength; the circulation about an area of infarction may have a chance to become reëstablished so far as possible; the heart is submitted to the least possible strain while the softened area is becoming scarred. In instances of angina where the attacks are becoming more frequent, a rest treatment is often of great value not only in that it spares an exhausted heart unnecessary beats, but in that it gives the patient an invaluable opportunity to adjust himself to the proper manner of life.

Under such circumstances what does one mean by rest? At what should we aim? Rest in bed at home? No. That is but a halfway measure. If it be possible the patient should be at rest in a hospital, wholly separated from his affairs, or if it must be at home, he should be isolated and under the care of a nurse. The patient almost always asks why home is not just as good as a hospital. Although he protests, it is usually not so very difficult to explain the situation. Few busy men can rest, really rest, at home. How many of us have tried to retire to the upper floors of our house and sought to spend a few days entirely freed from the cares of everyday life? How impossible it is! Every ring of the doorbell, every rattle of the telephone, suggests forgotten duties. The moment we are left alone we desire to get out of bed to arrange this or that little thing which must be done before the rest really begins, and the rest never comes. More than that, at home one has a sort of a right, or at any rate feels a sense of duty to direct or advise or meddle with a thousand little things. In a hospital or, if impossible, so well as one can at home, the patient should be guarded from every interruption. He should be induced entirely to throw aside his business affairs. He should be kept absolutely in bed under rigid rules; and it seems to me that the importance of rigid rules, such as forbidding him to rise from bed even to use the commode, is as important here as in any so-called "rest cure." The value of such rigid, martinet-like rules in the care of such a patient at the beginning, which is obvious in the instance of a grave coronary thrombosis, lies in the circumstance that it impresses deeply upon the patient the necessity of care in the future. At the same time, the improvement which usually follows the rest encourages him and gives him hope. There is no manner in which one may so certainly induce the patient to lead the proper life in the future as by a rigid period of rest and retraining. The period of rest after a severe coronary thrombosis may have to be very long, and it is often wise to keep a patient who has had merely persistent anginoid symptoms in bed for at least a month and then to give another month in very, very gradual retraining. While in bed it is important that the patient should have thorough general massage so as to keep the muscles in the best possible condition. When one begins to allow the invalid to sit up and get out of bed the progress should be step by step. A month's rest in bed in a hospital demands nearly a month of retraining and graded exercises before he leaves, and, where it is possible, I always like to send the patient for three weeks or a month thereafter to a good sanatorium where he may be under the care of well-trained men—a sanatorium like Clifton Springs, for instance—or to a resort like Atlantic City, so that he may get back into the habits of a normal life under proper observation. The permanency of the improvement following such treatments is sometimes astonishing, not only in those patients who have had a definite coronary thrombosis, but sometimes in instances where the anginoid attacks have lasted for several years and have given every promise of pursuing a progressive course.

My friend X, aged forty-eight, an engineer with important responsibilities, had begun, in 1913, to suffer from a sense of tightness across the front of the chest on effort. In the fall of 1914 he consulted me because the attacks, brought on by slight effort or emotion, had become very severe. They were located behind the sternum, were like a "red-hot iron" and radiated down his left arm and to a lesser extent his right. The pressure rose during attacks. A long rest, first in a hospital, then at Clifton Springs and in the country, with gradual retraining, was followed by a complete disappearance of the attacks. The patient learned how to live. He resigned his position, but soon was able to take up work as a consulting engineer and is today, after fifteen years, an active, successful man. He has resumed golf in moderation. He feels sure that violent effort would bring on his pains, but he has learned his lesson, and while, fifteen years ago, slight effort brought on severe attacks in bed, today he is leading a useful life.

My colleague, B. H. Rutledge, has recently had charge of a man of over seventy, who had had

characteristic angina of effort, of increasing frequency for eight years, so bad that they came on under most trivial effort, and waked him repeatedly at night. A treatment of complete rest and retraining lasting three months has, for the time being, wholly ended the pains. This man has resumed his business successfully for nearly a year; he has not had an attack for a year.* Cured? Of course not, but greatly benefited and enabled to live a comfortable life which may endure for a considerable period.

Medically we know only palliatives, but our general management of the case may bring about practical recovery for considerable periods of time. There is no condition where the skill and judgment of the physician comes into greater play.

As I have said elsewhere, the management of the family is the most difficult problem. The patient is usually one's best confidant. The family are very hard to deal with and it is in their power to make the patient's life utterly miserable. One must seek by every conceivable means to induce the family to let the patient alone, and never, by word or act, to show their anxiety. To do this is not always possible. Too often a loving but ill-balanced wife or husband, by constant manifestations of anxiety, may ruin the life of the patient.

At the outset of these rambling remarks I spoke of the tyranny of words under which we all live. The tyranny of the slogan or the shibboleth, while it may be a humiliating evidence of human weakness and impressionability, is, at the same time, a striking example of the power of words. Sometimes, I think, we physicians forget that if the knife be the most valuable implement of the surgeon, so is the tongue the most precious instrument of the physician. There are still relatively few specifics in medicine. It is by our counsel, by our moral influence, by our powers of explanation or illustration or reasoning, that we induce the patient to realize that which he must do to preserve himself and others from disaster. It is by the tongue that we achieve our chief results. Without careful education and training, without a good head to begin with, without experience and the power to profit by experience, no surgeon can properly use his knife; no physician can properly use his tongue. There is no regular rule by which the physician may be guided. Medicine, while we remain human beings, can never be practised by rule; if it could, the function of the physician would be much easier if, indeed, it continued to exist. There is no specific for that syndrome which we call "angina pectoris," but there are few maladies which can be more profoundly influenced by the wise counsel of a judicious physician. It is easier, far easier, to sit down and write a prescription which may be handed to the patient with a few words of direction in an instance of tertian malaria, than it is to guide a patient with early anginoid symptoms into that course of life which may enable him to play his full part in the world's activities. But the results in the latter instance may be just as great, if harder to achieve.

Let us beware of the tyranny of words, but let us not forget the power of words; for in wise words, wisely used, lies a great part of our art.

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*It is now nearly two years. The patient remains active and in good condition.

REFERENCE

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ACUTE CHOLECYSTITIS—ITS SURGICAL TREATMENT*

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THE treatment of acute cholecystitis varies markedly. On the Continent the treatment is essentially radical, in America it is mainly conservative, but within each of these areas there are widely divergent views on the subject. The study here presented was undertaken at the San Francisco Hospital to establish the status of the treatment of acute cholecystic disease in the San Francisco Bay region of California.

CASES IN THE LITERATURE

Hotchkiss [1] in 1894 reported the first case of acute gangrenous cholecystitis. In 1904 Mayo-Robson [2] reported two cases, and in 1906 Ross [3] reported five cases and gathered eleven from the literature. Since that time scattered case reports have been published by Tate,[4] Whitacre,[5] Cramp,[6] Cottam,[7] Andrew,[8] Cameron,[9] Ferguson,[10] Gould and Whitby,[11] and others (Table 1). To interpret the literature on this subject it is necessary to understand the different writers' conceptions of acute gangrenous cholecystitis. Unfortunately this is difficult because of the variations in classification and the personal element in interpretation of the pathology of acute cholecystitis.

CLASSIFICATION OF GALL-BLADDER LESIONS

I have tried to follow MacCarty's [12] classifications of gall-bladder lesions, considering acute cholecystitis as simple "acute catarrhal cholecystitis" and "cholecystitis purulenta necrotica." [13] The admissions into the San Francisco Hospital, under the heading of acute cholecystitis, include acute catarrhal cholecystitis, acute exacerbations of chronic cholecystitis, the acute cholecystitis of pregnancy, acute hydrops, acute empyema, acute phlegmonous cholecystitis, and acute gangrenous cholecystitis (Table 2).

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